CARDIOVASCULAR MEDICINE

APOE alleles are not associated with calcific aortic stenosis

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Objectives: To analyse the association of APOE alleles with aortic stenosis (AS) in a large study population.
Methods: Patients with AS (n = 538) and a control group of the same age without heart disease (n = 536) were recruited. Left heart catheterisation was performed and mean gradient, aortic valve area, presence of stenotic coronary artery disease (CAD) and cardiovascular risk factors (hypercholesterolaemia, hypertension, smoking, diabetes mellitus and family history of CAD) were assessed. The frequency of the APOE major alleles e2, e3 and e4 was assessed by genotyping the polymorphisms APOE334 and APOE472 with a 5′ exonuclease assay (TaqMan).
Results: Mean gradient across the aortic valve in cases was 50 (SD 20) mm Hg corresponding to a mean aortic valve area of 0.84 (SD 0.34) cm^2. 270 patients with AS had stenotic CAD. Among patients with AS, the prevalence of hypercholesterolaemia (64% *v* 40%, $p < 0.001$), smoking (43% *v* 27%, $p < 0.001$), diabetes (27% *v* 17%, $p < 0.01$), family history of CAD (30% *v* 21%, $p \leqslant 0.05$), and male sex (65% *v* 44%, $p < 0.001$) was higher in those with than in those without CAD. The frequency of the major alleles was not different between cases and controls (APOE e2: 104 (19.3%) *v* 94 (17.5%); APOE e3: 319 (59.3%) *v* 332 (61.9%); APOE e4: 115 (21.3%) *v* 110 (20.5%); all $p > 0.10$).
Conclusion: APOE e4 is not associated with AS, reflecting the different genetic backgrounds of CAD and AS.

The genetic disposition for coronary artery disease (CAD) and myocardial infarction (MI) is known, but that for degenerative calcific aortic stenosis (AS) has been less intensely investigated.[1 2] Some case reports have suggested that AS is heritable.[3–7] Whereas the heritability of MI, atherosclerosis of the abdominal aorta, aortic root size and bicuspid aortic valve seem probable, however, the heritability of AS is uncertain.[1 8–10] Previous association studies have linked genetic polymorphism of the VDR gene and inflammatory genes to AS.[11 12]

CAD and AS share many similarities and the pathogenesis of AS has been linked to cardiovascular risk factors. Many patients with AS have these risk factors,[13 14] which may be present because of the overlap with CAD, as cardiovascular risk factors are always associated with CAD but not always with AS.[15] Of the cardiovascular risk factors, hypercholesterolaemia is of special interest because early valvular lesions have deposits of cholesterol.[16] In some retrospective trials calcification was reduced by statin treatment, but in a randomised prospective trial intensive lipid-lowering treatment did not halt the progression of AS or induce its regression.[17–20]

Patients who are homozygous for familial hypercholesterolaemia, however, sometimes have severely calcified aortic roots and valves.[21 22] Thus, cholesterol is likely to be a key player in the pathogenesis of AS. APOE, with its major alleles e2, e3 and e4, is the key candidate gene, because apolipoprotein E is an essential structural component of cholesterol and is expressed in diseased valves.[23] So far, one study with 62 patients and 62 controls found a higher prevalence of the e2 allele in patients with AS. A second study with 43 patients and 759 controls found a higher prevalence of the e4 allele.[24 25] The present study was conducted to analyse the association of the APOE alleles e2, e3 and e4 with AS in a larger study population.

METHODS

This study has been carried out in accordance with the principles of the Declaration of Helsinki and was approved by the local ethics committee of the University Hospital of Aachen. According to the local ethics committee statement all phenotype data and blood samples were made anonymous before genotype–phenotype relationships were analysed. All participants were Caucasian.

AS cohort: cases and controls

Consecutive patients with AS diagnosed by echocardiography who were admitted for left heart catheterisation formed the AS case population. The patients were recruited. Results of the first 187 patients were published previously.[12] There is no overlap with two earlier studies of our group.[11 15] Inclusion criteria were first elective diagnostic coronary angiography. Left heart catheterisation was performed because of anticipated heart valve surgery or to define the severity of AS if non-invasive testing was inconclusive. Therefore, the patients constituted a selected population with severe AS. Exclusion criteria were mild or moderate aortic stenosis, severe aortic regurgitation, a history of endocarditis, and clear evidence of bicuspid valves, rheumatic fever or other relevant valvular disease. Of several thousand consecutive patients with clinically suspected CAD (because of chest discomfort or positive stress test) referred for coronary angiography who were older than 65 years, those with no evidence of structural heart disease (no stenotic CAD, no wall motion abnormalities, no reduced left ventricular function, a mean gradient < 5 mm Hg across the aortic valve and no other relevant

Abbreviations: AS, degenerative calcific aortic stenosis; CAD, coronary artery disease; MI, myocardial infarction

Table 1 Overview of age, risk profile and APOE allele frequencies in cases (patients with degenerative calcific AS) and controls (no heart disease, >65 years old) characterised by left heart catheterisation

	Cases			
	All	With CAD	Without CAD	Controls
Number	538	270	268	536
Stenotic CAD	270 (50%)	270 (100%)	0	0
ΔP AV (mm Hg)	50 (20)‡	47 (18)‡	53 (22)‡	<5§
AVA (cm^2)	0.84 (0.34)¶	0.88 (0.36)¶	0.80 (0.31)¶	NA
Age (years)	72 (6)	72 (6)	71 (7)	71 (5)
Men	292 (54%)	175 (65%)***	117 (44%)	215 (40%)
Women	246 (46%)	95 (35%)***	151 (56%)	321 (60%)
Body mass index (kg/m^2)	26.58 (3.83)	26.29 (3.32)	26.86 (4.28)	26.50 (3.73)
Cardiovascular risk factor				
Hypercholesterolaemia	282 (52%)	174 (64%)***	108 (40%)	305 (57%)†
Hypertension	362 (67%)	189 (70%)	173 (65%)	351 (66%)
Smoking	190 (35%)	117 (43%)***	73 (27%)	115 (22%)††
Diabetes mellitus	120 (22%)	74 (27%)**	46 (17%)	87 (16%)††
Family history of CAD	136 (25%)	81 (30%)*	55 (21%)	132 (25%)
Genotype				
APOE 334 TT	402 (75%)	207 (77%)	195 (73%)	412 (77%)
APOE 334 TC	129 (24%)	58 (22%)	71 (27%)	114 (21%)
APOE 334 CC	7 (1%)	5 (2%)	2 (1%)	10 (2%)
APOE 472 CC	434 (81%)	219 (81%)	215 (80%)	442 (83%)
APOE 472 CT	88 (16%)	44 (16%)	44 (16%)	91 (17%)
APOE 472 TT	16 (3%)	7 (3%)	9 (3%)	3 (1%)
APOE e2	104 (19%)	51 (19%)	53 (20%)	94 (18%)
APOE e3	319 (59%)	162 (60%)	157 (59%)	332 (62%)
APOE e4	115 (21%)	57 (21%)	58 (22%)	110 (21%)
APOE e2‡‡	89 (17%)	46 (17%)	43 (16%)	80 (15%)
APOE e3‡‡	319 (59%)	162 (60%)	157 (59%)	332 (62%)
APOE e4‡‡	130 (24%)	62 (23%)	68 (25%)	124 (23%)

*p ⩽ 0.05, **p ⩽ 0.01, ***p ⩽ 0.001, patients with AS and coronary artery disease (CAD) *v* AS without CAD; †p ⩽ 0.05, ††p<0.001, patients with AS and CAD *v* controls; ‡determined in only 517 of 538 patients (258 of 270 and 259 of 268); §patients with a mean gradient across the aortic valve ⩾5 mm Hg were excluded; ¶determined in only 412 of 538 patients (204 of 270 and 208 of 268); ††gene carriers with e2/4 categorised as APOE e4.
ΔP AV, mean gradient across the aortic valve determined by pull back; AVA, aortic valve area calculated by the Gorlin formula; CAD, stenotic (diameter stenosis >50% in at least one vessel) coronary artery disease; NA, not assessed.

cardiac diagnosis such as pericarditis or tachycardia) formed the control population.

Phenotyping

Cardiovascular risk factors were defined as hypercholesterolaemia (cholesterol concentration > 5.18 mmol/l or medically treated), arterial hypertension (blood pressure > 140/90 mm Hg or medically treated), diabetes mellitus (overnight fasting serum glucose > 6.99 mmol/l on at least two occasions or medically treated), family history of CAD (one first-degree relative with CAD/MI) and smoking (regular smoking habit). All patients underwent coronary angiography in at least four views of each coronary artery. Stenotic CAD was defined as a diameter stenosis of > 50% in at least one vessel. Two experienced interventional cardiologists analysed the angiograms independently by visual interpretation. The gradient across the aortic valve was measured by pull back of the catheter from the left ventricle into the ascending aorta. The mean gradient was calculated by a computer-assisted program (Metek, Roetgen, Germany). If right heart catheterisation was performed the aortic valve area was calculated with the Gorlin formula.

Genotyping

From EDTA whole blood, genomic DNA was isolated from leucocytes with the Invisorb Spin Blood Mini Kit (InVitek, Berlin, Germany). APOE gene sequences were amplified by polymerase chain reaction with a Hybaid thermocycler. Genotyping was done by allele-specific oligonucleotide probes in a procedure combining polymerase chain reaction and 5′ nuclease reaction with specific primers. APOE e2, e3 and e4 alleles (determined by APOE 334 T/C and APOE 472 C/T polymorphisms) were identified by the TaqMan technique in the ABI PRISM sequence system with fluorescence-marked probes as previously described.[26] Some studies have excluded people with the 2/4 genotype. However, we did not exclude these patients because in a pilot study with over 2000 healthy young men we found a comparable cholesterol concentration in those with genotype 2/4 and 2/2 or 2/3. We therefore categorised 2/4 as e2. However, categorising 2/4 as e2 is controversial and, in addition, we reanalysed the data by categorising 2/4 (n = 15 in cases, n = 14 in controls) as e4. Both results are presented. Hardy–Weinberg equilibrium was tested.

Table 2 Significant association of APOE alleles with presence of hypercholesterolaemia in cases (patients with degenerative calcific aortic stenosis) and controls (no heart disease, >65 years old)

	APOE allele		
	e2	e3	e4
Cases (n = 538)			
Number	104	319	115
Hypercholesterolaemia	45 (43%)	166 (52%)	71 (62%)*
Controls (n = 536)			
Number	94	332	110
Hypercholesterolaemia	49 (52%)	179 (54%)	77 (70%)**

*p = 0.023; **p = 0.008.

Statistical analysis

Data were statistically evaluated with SPSS for Windows V.12.0 software (SPSS Inc, Chicago, Illinois, USA) by applying χ^2 and cross-tabs analysis. A double-sided $p < 0.05$ was considered to be significant. Quantitative data were given as mean (SD) and qualitative data as frequencies. The null hypothesis was that the frequencies of APOE alleles would not differ between patients with AS and controls.

RESULTS

Study population

As table 1 shows, the study population consisted of older patients with severe aortic stenosis.

Cardiovascular risk factors

Table 1 shows that patients with stenotic CAD in addition to AS had a higher prevalence of cardiovascular risk factors such as hypercholesterolaemia, diabetes mellitus, positive family history, smoking and male sex than did patients with AS but without stenotic CAD. Similarly, compared with patients with AS and CAD, fewer controls had hypercholesterolaemia, smoking and diabetes.

APOE alleles

Hardy–Weinberg equilibrium was present in the whole study population (APOE 334: observed TT 814 TC 243 CC 17 *v* expected TT 816 TC 241 CC 17, $p = 0.99$; APOE 472: observed CC 876 CT 179 TT 19 *v* expected CC 869 CT 194 TT 11, $p = 0.25$). The APOE allele frequencies of patients with AS were not different from those without structural heart disease at the same age. However, as table 2 shows, APOE alleles were significantly associated with the presence of hypercholesterolaemia in both the case and control populations.

DISCUSSION

The association of certain genetic polymorphisms with AS remains a very interesting issue with the intriguing possibility of innovative therapeutic and preventive strategies. However, results so far have been limited. Hypercholesterolaemia is of interest because cholesterol is a component of early valvular lesions.[16] In cholesterol metabolism APOE is an especially interesting candidate gene because it has been found in diseased valves.[23] Two recent trials found that the functionally important APOE e2, e3 and e4 alleles are associated with AS.[24 25] The purpose of our study was to reproduce these findings in a larger group of patients. The major finding of our study is that APOE e2, e3 and e4 genotypes were not associated with AS. This is in contrast with the two smaller, previously published trials.[24 25] Our cohort of patients with AS is the largest investigated so far. Defining a control group is always difficult and subject to criticism. Whereas Avakian *et al*[24] approached this problem by matching 62 controls from a healthy population with 62 cases, Novaro *et al*[25] selected 759 patients with other heart diseases (predominantly CAD, MI and other valvular heart disease) as controls matched with 43 cases. We have deliberately favoured a control group in which CAD, MI and valvular heart disease were clearly excluded. Because patients with AS are older, an appropriate control group must also be older. However, the high prevalence of known and unknown heart disease in the general population older than 65 confers a high risk of false selection. We selected patients who had heart disease excluded by heart catheterisation. We are aware that this also caused a certain selection bias, such as a high prevalence of cardiovascular risk factors, which might have affected the indication for heart catheterisation, but the prevalence of cardiovascular risk factors in the general elderly German population is high and often underestimated.[27 28]

Despite the problem of the overlap with CAD, however, we have two groups with two distinctly different and accurate phenotypes (severe AS *v* no AS), and accuracy of phenotypes is often claimed.[29]

Concerning hypercholesterolaemia, the APOE gene is an excellent candidate. In our study population APOE alleles were significantly associated with the prevalence of hypercholesterolaemia, indicating a stable genotype–phenotype relationship. APOE alleles are functionally relevant and are known to be associated with CAD.[30–34] This study was not conducted to analyse an association with MI or CAD. For this purpose the study population is too old, because genetic factors are associated with MI at a premature age (< 65 years), whereas in older people genetic factors apparently do not have a major role in MI.[1] In our opinion this population is therefore not suitable for the detection of genetic factors associated with premature, genetically triggered MI and no conclusion about the association of APOE and CAD should be drawn from this study.

We might have missed a small effect but, given our data in the light of the two previous small studies, we think that no association of APOE alleles with AS can be claimed.[24 25] The lack of association may reflect that AS and CAD are two different diseases with different genetic backgrounds. In a previous study, in which DNA was not available, we found no association of hypercholesterolaemia with AS, whereas hypercholesterolaemia was associated with CAD in this and the previous study. AS and CAD share many features, but hypercholesterolaemia may not be linked to AS in the same way as to CAD. This speculation may be endorsed by the lack of a therapeutic effect of lipid-lowering treatment in AS.[20]

The limitation of our study is the lack of prospective data.

We conclude that, in contrast with their association with CAD, APOE alleles are not associated with AS, reflecting the different genetic bases of AS and CAD. Cholesterol remains of interest for the pathogenesis of AS because it is found in valvular lesions.[16 35] However, the role of cholesterol may be different in the calcification process of AS and CAD. Other genetic (or non-genetic) factors such as calcium or inflammatory systems may be important for the disease and should be investigated in further trials.

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IMAGES IN CARDIOLOGY

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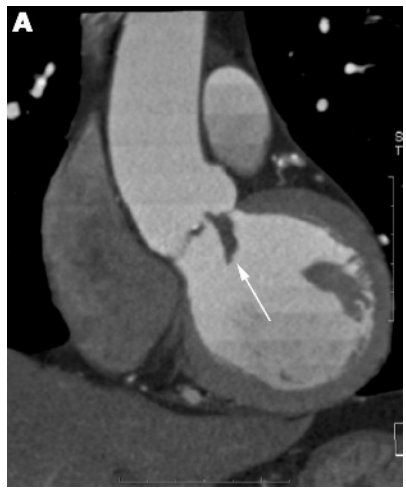
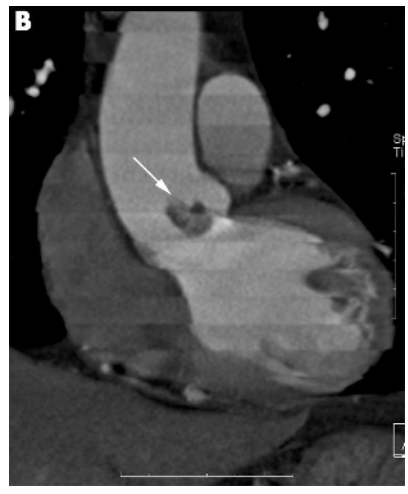
Aortic valvular endocarditis visualised by 16-row detector multislice computed tomography

A 58-year-old man was admitted to our institution for a left-sided homonymous hemianopsia with fever and a systolic murmur. Blood cultures were positive for *Enterococcus faecalis* and an ischaemic stroke in the left posterior cerebral artery territory was observed on CT examination. Echocardiography disclosed an extensive vegetation on the aortic valve and a mild aortic insufficiency. A 16-row detector MSCT (Sensation 16 Siemens) with ECG gating demonstrated the aortic vegetation (10 × 23 mm) on the ventricular side of the aortic valve (panel A) with systolic protrusion in the aortic root (panel B). Intravenous antibiotics were immediately started. Because of the stroke, resection of the vegetation and subsequent valve repair were delayed four weeks after the initial diagnosis, without recurrent embolic event. In the absence of coronary artery stenoses on the cardiac CT imaging, and because of the length of the vegetation, a preoperative coronary angiography was not performed. To our knowledge this is the first case of aortic endocarditis visualised by 16-row MSCT managed with a totally non-invasive approach before surgery.



Sixteen-row detector multislice CT of a valvular aortic endocarditis: large vegetation (23 × 10 mm, arrow) on the ventricular side of the left anterior cusp in diastole.



Aortic protrusion of the vegetation during systole.

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